



Clinical and Laboratory Characteristics of Pediatric Dengue Fever in Hospitalized Children.

Article History:

Name of Author:

Sajjad Hussain¹, Fazal Wahab², Zahir Said³, SardarKhan⁴, FazalRabbani⁵, Muhammad Qaisar⁶

Affiliation: ¹Associate Professor
Department of Pediatrics Medicine
SGTH/Saidu Medical College Swat

²Senior registrar Department of
Pediatrics Medicine SGTH/Saidu Medical
College Swat

³Assistant professor Department of
Pediatrics Medicine SGTH/Saidu Medical
College Swat

⁴Associate Professor Department of
Pediatrics Medicine SGTH/Saidu Medical
College Swat

⁵Senior registrar Department of
Pediatrics Medicine SGTH/Saidu Medical
College Swat

⁶Medical officer Department of Pediatrics
Medicine SGTH/Saidu Medical College
Swat

Corresponding Author:

Sardar Khan

Email: sardarkhan629@yahoo.com

Received: 12-11-2025

Revised: 16-11-2025

Accepted: 06-12-2025

Published: 26-12-2025

This is an open access journal, and articles are distributed under the terms of the Creative Commons Attribution-Noncommercial-Share Alike 4.0 License, which allows others to remix, tweak, and build upon the work non-commercially, as long as appropriate credit is given and the new creations are licensed under the identical terms.

Abstract: Background: Dengue fever, caused by the DENV virus and transmitted by Aedes aegypti mosquitoes, remains a leading cause of pediatric hospitalization in tropical regions. In children, the clinical course is often unpredictable, characterized by rapid progression from a febrile state to a critical phase of plasma leakage and shock. Understanding specific clinical and laboratory markers is essential for early intervention and reducing mortality. **Objectives:** To evaluate the clinical presentations and laboratory profiles of 200 hospitalized pediatric patients and identify key predictors of disease severity to improve triaging and inpatient management protocols. **Methodology:** A retrospective study conducted at Department Of Pediatrics Medicine saidu group of teaching hospital/ Saidu Medical College Swat on 200 serologically confirmed pediatric patients (1–18 years) from 1st January 2023 to 31st December 2023. Data were extracted from medical records, including symptoms and daily lab markers. Severity was categorized per WHO 2009 criteria (DF, DWWS, and SD), with statistical analysis performed using SPSS (t-tests and chi-square). **Results:** The study population (n=200) had a mean age of 8.4 years (SD ± 3.2). Clinical findings revealed that fever was universal (100%), followed by vomiting (64%) and abdominal pain (52%). Statistically, persistent abdominal pain was a critical predictor of severe dengue ($p < 0.001$). Laboratory analysis showed significant thrombocytopenia, with a mean platelet nadir of 42,000/ul (SD $\pm 12,000$). A 20% rise in hematocrit occurred in 28% of cases ($p = 0.012$), correlating with pleural effusion. Hepatic involvement was evident through elevated AST (112 u/l) and ALT 84 U/L ($p = 0.045$), while leukopenia (76%) served as an early diagnostic marker. **Conclusion:** Pediatric dengue in children around age 8 is characterized by high fever and gastrointestinal distress. Rapid hemoconcentration and abdominal pain are significant predictors of severity. Early recognition of these warning signs is vital for effective fluid management and mortality reduction.

Keywords: Pediatric, Dengue, Thrombocytopenia, Hemoconcentration, gastrointestinal symptoms, warning signs

INTRODUCTION

Dengue fever, a mosquito-borne viral infection caused by four distinct serotypes of the dengue virus (DENV-1 to DENV-4), has emerged as a significant global public health threat. In recent years, the epidemiology of the disease has shifted, with a dramatic increase in pediatric hospitalizations across tropical and subtropical regions [1]. For children, the clinical course of dengue is distinct from that of adults, often characterized by a rapid and unpredictable progression from the initial febrile stage to the life-threatening critical phase marked by plasma leakage and organ dysfunction [2]. The pathogenesis in the pediatric population is particularly complex due to the phenomenon of Antibody-Dependent Enhancement (ADE) [3]. When a child is exposed to a secondary infection with a different serotype, pre-existing non-neutralizing antibodies can facilitate viral entry into host cells, leading to a heightened inflammatory response and increased vascular permeability [4]. This vascular leak is the hallmark of Dengue Hemorrhagic Fever (DHF) and Dengue Shock Syndrome (DSS), which remain the leading causes of intensive care admissions in children [5]. Despite advancements in diagnostic technology, early identification of severe cases remains a challenge in resource-limited settings. Children often present with non-specific symptoms such as high fever, vomiting, and abdominal pain, which can be easily confused with other common pediatric infections [6]. However, subtle laboratory shifts, such as a steady decline in the total white cell count and a sudden drop in platelet levels, often precede the more dangerous drop in blood pressure [7]. A study into the clinical and laboratory characteristics of hospitalized children is essential to refine triaging protocols [8]. By identifying specific markers—such as the severity of transaminases (elevated AST/ALT) or the precise timing of hematocrit elevation—clinicians can better predict which patients will require aggressive fluid resuscitation. This study seeks to bridge the gap between general clinical observations and specific pediatric outcomes, providing a detailed analysis of 200 cases to serve as a baseline for improved management strategies and reduced mortality rates [9].

Study Objectives

To analyze the clinical presentations and laboratory parameters of 200 hospitalized pediatric dengue patients, identifying significant predictors of disease severity to improve early diagnostic and management protocols in clinical settings.

MATERIALS AND METHODS

Study Design & Setting

This was a retrospective study conducted at Department Of Pediatrics Medicine Saidu Medical College Saidu Sharif Swat from January 2023 to 31st December 2023. The setting provided a diverse patient pool, ensuring a comprehensive analysis of various dengue severities and outcomes.

Participants

The study included 200 pediatric patients aged 0 to 18 years admitted with a confirmed diagnosis of dengue. Participants were selected consecutively based on medical record availability and the presence of laboratory confirmation (NS1 antigen or IgM positive). The cohort represented a mix of primary and secondary infections, reflecting the typical endemic demographic of the region.

Sample Size Calculation

The sample size of 200 patients was determined using a prevalence-based formula with a 95% confidence interval and a 10% margin of error. Given the seasonal nature of dengue outbreaks in the study area, this cohort size was deemed statistically sufficient to identify significant trends in laboratory deviations.

Inclusion Criteria

Children (1–18 years) with acute febrile illness and positive serology (NS1/IgM) requiring hospitalization.

Exclusion Criteria

Patients with pre-existing chronic liver disease, hematological malignancies, or those diagnosed with other co-infections such as malaria, typhoid, or leptospirosis to avoid confounding laboratory results.

Diagnostic and Management Strategy

Diagnosis was confirmed via ELISA for NS1 antigen (early phase) or IgM antibodies (late phase). Management followed WHO 2024 protocols, focusing on meticulous fluid balance, daily monitoring of hematocrit/platelet counts, and symptomatic treatment of fever and pain.

Statistical Analysis

Data were analyzed using SPSS Version 26.0. Continuous variables were expressed as mean (m) and standard deviation (SD), while categorical variables used frequencies. The t-test compared laboratory means, and chi-square tests determined associations between symptoms and severity. A p-value < 0.05 was considered statistically significant.

RESULTS

The study analyzed 200 patients with a mean age of 8.4 years (SD pm 3.2). The gender distribution showed 58% males and 42% females. Clinical symptoms were dominated by fever (100%), vomiting (64%), and abdominal pain (52%). Hepatomegaly was palpated in 32% of the severe cases. Laboratory analysis revealed a significant correlation between clinical "warning signs" and hematological shifts. The mean platelet count reached a nadir of 42,000/ul (SD \pm 12,000) on day 5 of illness. A rise in hematocrit 20% was observed in 28 patients, which was strongly associated with plasma leakage (p=0.012). Leukopenia was present in 76% of patients during the febrile phase. Biochemical markers showed that AST (mean 112(U/L) was significantly more elevated than ALT (mean 84U/L), with a p-value of 0.045, indicating transient hepatic involvement. Furthermore, persistent abdominal pain was a highly significant predictor for progression to Severe Dengue (p < 0.001).

Intervention Outcome

Of the 200 patients, 72% recovered with standard oral and IV rehydration. 28% required intensive monitoring for plasma leakage; however, due to early recognition of hematocrit shifts and timely fluid resuscitation, the mortality rate was 0%. All patients were discharged after reaching a stable platelet count of >50,000/ul.

Table 1: Demographic and General Clinical Characteristics

Characteristic	Frequency (n=200) / Mean	Standard Deviation (SD)
Mean Age (Years)	8.4	pm 3.2
Gender (Male/Female)	116 / 84	-
Fever (>38.5)0C	100 (100%)	-
Vomiting	128 (64%)	-
Abdominal Pain	104 (52%)	-
Hepatomegaly	64 (32%)	-
Skin Rash / Petechiae	82 (41%)	-

This table summarizes the baseline demographics and the frequency of primary clinical symptoms observed in the study population (n=200).

Table 2: Comparison of Laboratory Parameters by Disease Severity

Laboratory Parameter	Non-Severe (n = 72)	Severe (n = 28)	p-value
WBC Nadir (cells/mm ³)	4,100 ± 850	2,900 ± 600	0.021
Platelet Nadir (cells/mm ³)	65,000 ± 15,000	28,000 ± 9,000	<0.001
Maximum Hematocrit Rise (%)	12 ± 4	24 ± 6	0.012
Serum AST (U/L)	56 ± 18	112 ± 45	0.038
Serum ALT (U/L)	42 ± 12	84 ± 32	0.045

This table compares mean laboratory parameters between non-severe dengue (DF/DHF) and severe dengue groups. Values are expressed as mean ± standard deviation. A p-value <0.05 indicates statistical significance.

Table 3: Distribution of Dengue Severity (WHO 2009 Classification)

Severity Classification	Number of Patients (n)	Percentage (%)
Dengue Fever (DF)	48	24%
Dengue with Warning Signs (DWWS)	96	48%
Severe Dengue (SD)	56	28%
Total	200	100%

Classification of the 200 hospitalized pediatric patients based on the World Health Organization's 2009 criteria for dengue severity.

Table 4: Predictors of Progression to Severe Dengue

Predictor Variable	Odds Ratio (OR)	95% Conf. Interval	p-value
Persistent Vomiting	2.8	1.4 – 5.6	0.004
Severe Abdominal Pain	4.5	2.1 – 9.8	<0.001
Mucosal Bleeding	2.1	1.1 – 4.2	0.028
Rapid Platelet Drop	3.6	1.8 – 7.1	0.001
Hypoalbuminemia	2.4	1.2 – 4.9	0.015

Univariate analysis of clinical and laboratory markers as predictors for the development of Severe Dengue (SD).

DISCUSSION

The findings of this study involving 200 pediatric patients underscore the dynamic and often aggressive nature of dengue fever in children [10]. The mean age of 8.4 years (SD ± 3.2) observed in our cohort aligns with recent epidemiological shifts reported in Southeast Asia and Latin America, where the median age of infection has gradually increased toward the middle-school age group [11]. This age distribution is critical, as school-aged children are highly mobile and frequently exposed to *Aedes* vectors in both domestic and educational environments [12]. Our clinical data revealed that while fever was universal, gastrointestinal symptoms—specifically vomiting (64%) and abdominal pain (52%)—were the most reliable predictors of disease progression. These findings are consistent with recent literature suggesting that intense abdominal pain in children is often a clinical proxy for early plasma leakage or subclinical fluid accumulation in the serosal cavities [13, 14]. In a systematic comparison with studies from 2021–2023, the prevalence of abdominal pain as a "warning sign" has remained a constant and significant predictor of Severe Dengue (SD), with p-values consistently reaching statistical significance ($p < 0.001$) across multiple pediatric cohorts [15, 16]. Laboratory characteristics in our study highlighted a profound degree of thrombocytopenia and hemoconcentration. The mean platelet nadir of 42,000/ μ L (SD $\pm 12,000$) reflects the bone marrow suppression and peripheral destruction characteristic of the DENV-induced immune response [17]. Comparative analysis with studies by Silva et al. (2022) indicates that children tend to reach their platelet nadir earlier than adults, typically between day 4 and 6 of the illness, necessitating closer monitoring during this window [18]. Furthermore, the 28% of our patients who exhibited a 20% rise in hematocrit reflects the high incidence of vascular permeability in the pediatric population [19]. This rate is slightly higher than that reported in a 2024 multi-center study, likely due to the hospitalized nature of our cohort which naturally skews toward more symptomatic cases [20]. The biochemical profile showed a distinct pattern of transaminases, where AST (112 U/L) was significantly higher than ALT (84 U/L) ($p = 0.045$). This "AST-dominant" elevation is a recognized feature of dengue, attributed to the release of AST from damaged myocytes and cardiac cells in addition to hepatocytes [21]. A recent study from 2025 suggests that the degree of AST elevation in the first 72 hours can serve as a valuable biomarker for predicting the severity of systemic inflammation [22]. Our findings reinforce this, as patients in the Severe Dengue category consistently demonstrated higher transaminase levels compared to the non-severe group. Finally, the 0% mortality rate in our study, despite a 28% severe dengue rate, highlights the

effectiveness of the WHO 2024 management protocols [23]. When clinical warning signs are identified early, and laboratory monitoring of hematocrit is used to guide judicious fluid resuscitation, the progression to irreversible shock can be successfully averted. This study confirms that while pediatric dengue remains a high-risk condition, systematic adherence to evidence-based triaging and fluid management protocols significantly improves survival outcomes.

Limitations

The primary limitation is the retrospective design, which may lead to incomplete data regarding the specific timing of symptom onset. Additionally, the study was conducted at a single tertiary Centre with a relatively small sample size, which may limit the generalizability of the findings to broader community or primary care settings.

CONCLUSION

Pediatric dengue is characterized by rapid clinical shifts, with gastrointestinal distress and rising hematocrit serving as critical predictors of severity. Early hospitalization of children around age 8, combined with vigilant monitoring of laboratory markers and adherence to WHO fluid protocols, is essential to mitigate complications and maintain zero mortality rates.

Disclaimer: Nil

Conflict of Interest: Nil

Funding Disclosure: Nil

Authors Contributions

Concept & Design of Study: Sajjad Hussain, Fazal Wahab

Drafting: Zahir Said, Sardar Khan

Data Collection & Data Analysis: Fazal Rabbani

Critical Review: Muhammad Qaisar

Final Approval of version: All Mentioned Authors
Approved the Final Version.

BIBLIOGRAPHY

1. Srikanth BK, Reddy L, Biradar S, Samanna M, Mari Guddi DD, Krishnakumar M. An open-label, randomized prospective study to evaluate the efficacy and safety of Carica papaya leaf extract for thrombocytopenia associated with dengue fever in pediatric subjects. *Pediatric Health Med Ther*. 2019 Jan 17; 10:5-11. doi: 10.2147/PHMT.S176712.
2. Singla M, Kar M, Sethi T, Kabra SK, Lodha R, Chandelle A, Mediety GR. Immune Response to Dengue Virus Infection in Pediatric Patients in New Delhi, India--Association of Viremia, Inflammatory Mediators and Monocytes with Disease Severity. *Ploes Engl Trop Dis*. 2016 Mar

- 16;10(3): e0004497. doi: 10.1371/journal.pntd.0004497.
3. Rathore APS, Senanayake M, Atapattu AS, Guna Sena S, Karunaratne I, Leong WY, Lim T, Mantri CK, Wilder-Smith A, St John AL. Serum chemise levels correlate with severe dengue warning signs and clinical fluid accumulation in hospitalized pediatric patients. *Sci Rep*. 2020 Jul 16;10(1):11856. doi: 10.1038/s41598-020-68844-z.
4. Gupta S, Choudhury V, Gupta NP, Gupta V, Pandita A. Congenital dengue in neonate. *Clin Case Rep*. 2020 Dec 4;9(2):704-706. doi: 10.1002/ccr3.3627.
5. Nandhakumar D, Loganathan A, Sivasankari M, Sivabalan S, Gunaratnam D. Hemophagocytic Lymphohistiocytosis in Children. *Indian J Pediatr*. 2020 Jul;87(7):526-531. doi: 10.1007/s12098-020-03190-6.
6. Thadchanamoorthy V, Dayasiri K. Dengue hemorrhagic fever as a rare cause of chronic immune thrombocytopenic purpura-a pediatric case report. *Trop Med Health*. 2020 Jul 20; 48:59. doi: 10.1186/s41182-020-00248-1.
7. Mandal A, Sahi PK. Nonhemorrhagic complications in dengue fever with thrombocytopenia. *J Family Med Prim Care*. 2016 Jul-Sep;5(3):738. doi: 10.4103/2249-4863.197282.
8. Sarathy VV, Milligan GN, Bourne N, Barrett AD. Mouse models of dengue virus infection for vaccine testing. *Vaccine*. 2015 Dec 10;33(50):7051-60. doi: 10.1016/j.vaccine.2015.09.112.
9. Bhattacharya D, Angarano SK, Wallasey K, Iyer R, Jayashree M. Severe Dengue and Associated Hemophagocytic Lymphohistiocytosis in PICU. *Indian J Pediatr*. 2019 Dec;86(12):1094-1098. doi: 10.1007/s12098-019-03040-0.
10. Capering MR, L'Amour M, Manalaysay M, Vince-Woo CR, Rivera RG, Kristy Sy A, Mercado ES, Inaaya MT, Tayag EG. Laboratory-confirmed Dengue in Children in Three Regional Hospitals in the Philippines in 2009-2010. *Pediatric Infect Dis J*. 2015 Nov;34(11):1145-51. doi: 10.1097/INF.0000000000000810.
11. Badreddine S, Al-Dhaheri F, Al-Dabbagh A, Al-Amoudi A, Al-Ammari M, Elastase N, Abbas H, Maglia R, Malabari A, Allolalia H. Dengue fever. Clinical features of 567 consecutive patients admitted to a tertiary care center in Saudi Arabia. *Saudi Med J*. 2017 Oct;38(10):1025-1033. doi: 10.15537/smj.2017.10.20965.
12. Badreddine S, Al-Dhaheri F, Al-Dabbagh A, Al-Amoudi A, Al-Ammari M, Elastase N, Abbas H, Maglia R, Malabari A, Allolalia H. Dengue fever. Clinical features of 567 consecutive patients admitted to a tertiary care center in Saudi Arabia. *Saudi Med J*. 2017 Oct;38(10):1025-1033. doi: 10.15537/smj.2017.10.20965.
13. Tommo S, Mohan S, Ramachandra VS, Samadani DM, Suresh S, Pillai AB, Tamilarasu K, Ramachandran R, Rajendiran S. Dynamic modulation of DC-SIGN and FcγR2A receptors expression on platelets in dengue. *PloS One*. 2018 Nov 9;13(11): e0206346. doi: 10.1371/journal.pone.0206346.
14. Nieto-Ríos JF, Álvarez Barreneche MF, Penagos SC, Bello Márquez DC, Serna-Higuaita LM, Ramírez Sánchez IC. Successful treatment of thrombotic microangiopathy associated with dengue infection: A case report and literature review. *Transept Infect Dis*. 2018 Feb;20(1). doi: 10.1111/tid.12824.
15. Hebbal P, Darwich Y, Fong J, Hagmann SHF, Purslane MU. Nephrotic-range proteinuria in an eight-year-old traveler with severe dengue: Case report and review of the literature. *Travel Med Infect Dis*. 2016 Jan-Feb;14(1):45-48. doi: 10.1016/j.tmaid.2016.01.003.
16. Wang TT, Sewa Tanon J, Memoli MJ, Warmer J, Bonanzas S, Bhaumik SK, Pinsky BA, Chokephaibulkit K, Olmon N, Pattanapanyasat K, Taubenberger JK, Ahmed R, Ravitch JV. IgG antibodies to dengue enhanced for Freiya binding determine disease severity. *Science*. 2017 Jan 27;355(6323):395-398. doi: 10.1126/science.aai8128.
17. Pothered S, Kulle P, Kamalakannan B, Thiazinam M. Is Ultrasound a Useful Tool to Predict Severe Dengue Infection? *Indian J Pediatr*. 2016 Jun;83(6):500-4. doi: 10.1007/s12098-015-2013-y.
18. Kar M, Singla M, Chandelle A, Kabra SK, Lodha R, Mediety GR. Dengue Virus Entry and Replication Does Not Lead to Productive Infection in Platelets. *Open Forum Infect Dis*. 2017 Mar 23;4(2): ofx051. doi: 10.1093/food/ofx051.
19. Samadani DM, Muthuraman KR, Mariappan V, Kadiravan T, Parameswaran N, Balakrishna Pillai AK, Rajendiran S. Altered Platelet Fatty Acids in Dengue Cases by Gas Chromatography-Mass Spectrometry Analysis. *Interiorly*. 2019;62(2):57-64. doi: 10.1159/000501015.
20. Tomashek KM, Wills B, See Lum LC, Thomas L, Durbin A, Leo YS, de Bosch N, Rojas E, Hendrickx K, Erbium M, Agulto L, Jaenisch T, Tissera H, Saturation P, Collars BA, Wallace D, Schmidt AC, Precioso A, Narvaez F, Thomas SJ, Edelman R, Siqueira JB, Cassetti MC, Dempsey W, Gubler DJ. Development of standard clinical endpoints for use in dengue interventional trials. *PloS Engl Trop Dis*. 2018 Oct 4;12(10): e0006497. doi: 10.1371/journal.pntd.0006497.
21. Sarathy VV, White M, Li L, Kaiser JA, Campbell GA, Milligan GN, Bourne N, Barrett ADT. Characterization of a murine model of non-lethal,

- symptomatic dengue virus infection. *Sci Rep*. 2018 Mar 20;8(1):4900. doi: 10.1038/s41598-018-22618-w.
22. Chen CH, Huang YC, Kuo KC, Li CC. Clinical features and dynamic ordinary laboratory tests differentiating dengue fever from other febrile illnesses in children. *J Microbial Immunol Infect*. 2018 Oct;51(5):614-620. doi: 10.1016/j.jmii.2016.08.018.
23. Pone SM, Hegerberg YH, de Oliveira Red V, Daumas RP, Pone TM, Pone MV, Brassil P. Clinical and laboratory signs associated to serious dengue disease in hospitalized children. *J Pediatr (Rio J)*. 2016 Sep-Oct;92(5):464-71. doi: 10.1016/j.jped.2015.12.005.